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NEWS 2	DEC 01	ChemPort single article sales feature unavailable
NEWS 3	JAN 06	The retention policy for unread STNmail messages will change in 2009 for STN-Columbus and STN-Tokyo
NEWS 4	JAN 07	WPIDS, WPINDEX, and WPIX enhanced Japanese Patent Classification Data
NEWS 5	FEB 02	Simultaneous left and right truncation (SLART) added for CERAB, COMPUAB, ELCOM, and SOLIDSTATE
NEWS 6	FEB 02	GENBANK enhanced with SET PLURALS and SET SPELLING
NEWS 7	FEB 06	Patent sequence location (PSL) data added to USGENE
NEWS 8	FEB 10	COMPENDEX reloaded and enhanced
NEWS 9	FEB 11	WTEXTILES reloaded and enhanced
NEWS 10	FEB 19	New patent-examiner citations in 300,000 CA/CAplus patent records provide insights into related prior art
NEWS 11	FEB 19	Increase the precision of your patent queries -- use terms from the IPC Thesaurus, Version 2009.01
NEWS 12	FEB 23	Several formats for image display and print options discontinued in USPATFULL and USPAT2
NEWS 13	FEB 23	MEDLINE now offers more precise author group fields and 2009 MeSH terms
NEWS 14	FEB 23	TOXCENTER updates mirror those of MEDLINE - more precise author group fields and 2009 MeSH terms
NEWS 15	FEB 23	Three million new patent records blast AEROSPACE into STN patent clusters
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NEWS 17	MAR 06	INPADOCDB and INPAFAMDB enhanced with new display formats

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 NEWS 18  MAR 11  EPFULL backfile enhanced with additional full-text
                  applications and grants
 NEWS 19  MAR 11  ESBIOBASE reloaded and enhanced
 NEWS 20  MAR 20  CAS databases on STN enhanced with new super role
                  for nanomaterial substances
 NEWS 21  MAR 23  CA/CAplus enhanced with more than 250,000 patent
                  equivalents from China
 NEWS 22  MAR 30  IMSPATENTS reloaded and enhanced
 NEWS 23  APR 03  CAS coverage of exemplified prophetic substances
                  enhanced
 NEWS 24  APR 07  STN is raising the limits on saved answers
 NEWS 25  APR 24  CA/CAplus now has more comprehensive patent assignee
                  information
 NEWS 26  APR 26  USPATFULL and USPAT2 enhanced with patent
                  assignment/reassignment information
 NEWS 27  APR 28  CAS patent authority coverage expanded
 NEWS 28  APR 28  ENCOMPLIT/ENCOMPLIT2 search fields enhanced
 NEWS 29  APR 28  Limits doubled for structure searching in CAS
                  REGISTRY

 NEWS EXPRESS JUNE 27 08 CURRENT WINDOWS VERSION IS V8.3,
              AND CURRENT DISCOVER FILE IS DATED 23 JUNE 2008.

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=> S (Kruppel-like factor 9) AND Treatment
L1          20 (KRUPPEL-LIKE FACTOR 9) AND TREATMENT

=> Dup Rem L1
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PROCESSING COMPLETED FOR L1
L2            14 DUP REM L1 (6 DUPLICATES REMOVED)
                 ANSWERS '1-3' FROM FILE MEDLINE
                 ANSWERS '4-13' FROM FILE CAPLUS
                 ANSWER '14' FROM FILE EMBASE

=> D ibib abs L2 1-14

L2   ANSWER 1 OF 14     MEDLINE on STN                  DUPLICATE 1
ACCESSION NUMBER:   2009226616     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 19036875
TITLE:              Stressor and glucocorticoid-dependent induction of the
                    immediate early gene kruppel-like
                    factor 9: implications for neural
                    development and plasticity.
AUTHOR:             Bonett Ronald M; Hu Fang; Bagamasbad Pia; Denver Robert J
CORPORATE SOURCE:   Department of Molecular, Cellular, and Developmental
                    Biology, The University of Michigan, Ann Arbor, Michigan
                    48109-1048, USA.
CONTRACT NUMBER:    1 R01 NS046690 (United States NINDS NIH HHS)
SOURCE:             Endocrinology, (2009 Apr) Vol. 150, No. 4, pp. 1757-65.
                    Electronic Publication: 2008-11-26.
                    Journal code: 0375040. E-ISSN: 1945-7170.
                    Report No.: NLM-PMC2659263 [Available on 04/01/10].
PUB. COUNTRY:       United States
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
                    (RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
                    (RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)
LANGUAGE:           English
FILE SEGMENT:       Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH:        200904
ENTRY DATE:         Entered STN: 25 Mar 2009
                    Last Updated on STN: 15 Apr 2009
                    Entered Medline: 14 Apr 2009
AB   Kruppel-like factor 9 (KLF9) is a
     thyroid hormone-induced, immediate early gene implicated in neural
     development in vertebrates.  We analyzed stressor and glucocorticoid
     (GC)-dependent regulation of KLF9 expression in the brain of the frog
     Xenopus laevis, and investigated a possible role for KLF9 in neuronal
     differentiation.  Exposure to shaking/confinement stressor increased
     plasma corticosterone (CORT) concentration, and KLF9 immunoreactivity in
     several brain regions, which included the medial amygdala and bed nucleus
     of the stria terminalis, anterior preoptic area (homologous to the
     mammalian paraventricular nucleus), and optic tectum (homologous to the
     mammalian superior colliculus).  The stressor-induced KLF9 mRNA expression
     in the brain was blocked by pretreatment with the GC receptor antagonist
     RU486, or mimicked by injection of CORT.  Treatment with CORT
     also caused a rapid and dose-dependent increase in KLF9 mRNA in X. laevis
     XTC-2 cells that was resistant to inhibition of protein synthesis.  The
     action of CORT on KLF9 expression in XTC-2 cells was blocked by RU486, but
     not by the mineralocorticoid receptor antagonist spironolactone.  To test
     for functional consequences of up-regulation of KLF9, we introduced a KLF9
     expression plasmid into living tadpole brain by electroporation-mediated
     gene transfer.  Forced expression of KLF9 in tadpole brain caused an
     increase in Golgi-stained cells, reflective of neuronal
     differentiation/maturation.  Our results support that KLF9 is a direct, GC
     receptor target gene that is induced by stress, and functions as an
     intermediary in the actions of GCs on brain gene expression and neuronal
     structure.

L2   ANSWER 2 OF 14     MEDLINE on STN                  DUPLICATE 2
ACCESSION NUMBER:   2006176887     MEDLINE
```

DOCUMENT NUMBER: PubMed ID: 16384861
TITLE: Progesterone receptor transactivation of the secretory leukocyte protease inhibitor gene in Ishikawa endometrial epithelial cells involves recruitment of Kruppel-like factor 9/basic transcription element binding protein-1.
AUTHOR: Velarde Michael C; Iruthayanathan Mary; Eason Renea R; Zhang Daying; Simmen Frank A; Simmen Rosalia C M
CORPORATE SOURCE: Department of Physiology and Biophysics, University of Arkansas for Medical Sciences and Arkansas Children's Nutrition Center, Little Rock, 72202, USA.
CONTRACT NUMBER: HD21961 (United States NICHD NIH HHS)
SOURCE: Endocrinology, (2006 Apr) Vol. 147, No. 4, pp. 1969-78. Electronic Publication: 2005-12-29. Journal code: 0375040. ISSN: 0013-7227.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, N.I.H., EXTRAMURAL) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200604
ENTRY DATE: Entered STN: 31 Mar 2006
Last Updated on STN: 15 Apr 2006
Entered Medline: 14 Apr 2006

AB Progesterone receptor (PR), a ligand-inducible transcription factor, mediates the physiological actions of progesterone (P) through two distinct isoforms, PR-A and PR-B, and numerous nuclear coregulators. We previously demonstrated that basic transcription element binding protein-1 (BTEB1), a transcription factor of the Kruppel-like family, is a functional PR-interacting protein, based on the subfertility phenotype and reduced P sensitivity of uterine PR target genes, on BTEB1 null mutation. Here we examined the role of BTEB1 in PR-mediated signaling in endometrial epithelial cells using Ishikawa human endocarcinoma cells and the P-responsive secretory leukocyte protease inhibitor (SLPI) gene. Treatment of Ishikawa cells with P for 24 h increased SLPI and BTEB1 transcript levels without similar effects on PR expression. P induction was abolished by the PR antagonist RU486, whereas knockdown of BTEB1 with short interfering RNA reduced P-responsive BTEB1 but not SLPI expression to basal levels. Forced expression of BTEB1, either by stable or transient transfections of BTEB1 expression constructs in endometrial carcinoma cells, enhanced SLPI promoter activity. Chromatin immunoprecipitation with anti-BTEB1 antibody demonstrated BTEB1 recruitment to the proximal GC-rich containing SLPI promoter region (-97 to -86) in human endometrial carcinoma (HecIA) cells overexpressing BTEB1. In Ishikawa cells, recruitment of BTEB1 to the proximal, GC-rich region and the distal, progesterone-responsive element-like containing region (-635 to -514) was P dependent and was accompanied by corecruitment of PR and the PR coactivator cAMP-response element binding protein-binding protein. PR-B, rather than PR-A isoform, preferentially associated with BTEB1 in the GC-rich region, whereas both PR isoforms were recruited to the progesterone-responsive element-like site along with BTEB1. Our findings define a novel pathway for BTEB1/PR cross-talk to facilitate P-dependent gene transcription in endometrial epithelial cells.

L2 ANSWER 3 OF 14 MEDLINE on STN
ACCESSION NUMBER: 2009285768 IN-PROCESS
DOCUMENT NUMBER: PubMed ID: 19375645
TITLE: GAR22: a novel target gene of thyroid hormone receptor causes growth inhibition in human erythroid cells.
AUTHOR: Gamper Ivonne; Koh Ki-Ryang; Ruau David; Ullrich Katrin; Bartunkova Jana; Piroth Daniela; Hacker Christine; Bartunek

Petr; Zenke Martin
CORPORATE SOURCE: Institute for Biomedical Engineering, Department of Cell Biology, RWTH Aachen University Medical School, Aachen, Germany.
SOURCE: Experimental hematology, (2009 May) Vol. 37, No. 5, pp. 539-548.e4.
Journal code: 0402313. E-ISSN: 1873-2399.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: NONMEDLINE; IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 21 Apr 2009
Last Updated on STN: 21 Apr 2009

AB OBJECTIVE: Thyroid hormone receptors (TRs) are ligand-dependent transcription factors with a major impact on erythroid cell development. Here we investigated TR activity on red cell gene expression and identified TR target genes. The impact of the TR target gene GAR22 (growth arrest-specific 2 [GAS2]-related gene on chromosome 22) on red cell differentiation was determined. MATERIALS AND METHODS: Stem cell factor/erythropoietin (SCF/EPO)-dependent red cell progenitors were differentiated in vitro in the presence or absence of thyroid hormone. Hormone-induced changes in gene expression were measured by a genome-wide approach with DNA microarrays. Ectopic expression of the TR target gene GAR22 was used to determine its impact on red cell differentiation. RESULTS: Ligand-activated TR effectively accelerated red cell progenitor differentiation in vitro concomitantly with inducing growth arrest. We demonstrate that activated TR-induced specific gene expression patterns of up- or downregulated genes, including distinct clusters associated with accelerated differentiation in response to treatment. Mining for T3-induced genes identified basic transcription element binding protein 1/Kruppel-like factor 9 (BTEB1/KLF9) and GAR22 as TR target genes. BTEB1/KLF9 is a known TR target gene while GAR22, initially identified as a putative tumor suppressor, represents a novel TR target gene. We demonstrate that ectopic GAR22 expression in red cell progenitors lengthens the cell cycle and causes growth inhibition, but leaves red cell gene expression unaffected. CONCLUSION: This study identifies GAR22 as a novel and direct TR target gene. Our results suggest that hormone-induced GAR22 might represent an important trigger of growth inhibition induced by thyroid hormone in red cell progenitors.

L2 ANSWER 4 OF 14 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2007:284115 CAPLUS
DOCUMENT NUMBER: 146:352574
TITLE: Double-stranded RNAs and their use for downregulating genes and treating cardiovascular diseases
INVENTOR(S): Chajut, Ayelet; Pinner, Elhanan
PATENT ASSIGNEE(S): Quark Biotech, Inc., USA
SOURCE: PCT Int. Appl., 145pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2007029249	A2	20070315	WO 2006-IL1036	20060906

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP,

KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN,
MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS,
RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ,
UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ,
CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH,
GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
KG, KZ, MD, RU, TJ, TM
EP 1933880 A2 20080625 EP 2006-796071 20060906
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL,
BA, HR, MK, RS
JP 2009507484 T 20090226 JP 2008-529781 20060906
PRIORITY APPLN. INFO.: US 2005-715414P P 20050909
US 2005-732188P P 20051031
WO 2006-IL1036 W 20060906

AB The invention relates to a double-stranded compound, such as siRNAs, which down-regulates the expression of one or more cardiovascular-related gene. The invention also relates to a pharmaceutical composition comprising the compound, or a vector capable of expressing the oligoribonucleotide compound, and a pharmaceutically acceptable carrier. The present invention also contemplates a method of treating a patient suffering from a cardiovascular disorder or other diseases comprising administering to the patient the pharmaceutical composition in a therapeutically ED so as to thereby treat the patient.

L2 ANSWER 5 OF 14 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2007:113586 CAPLUS
DOCUMENT NUMBER: 146:226597
TITLE: Gene expression profiles in esophageal cancer and their use in diagnosis, prognosis, therapy and drug design and selection
INVENTOR(S): Nakamura, Yusuke; Daigo, Yataro; Nakatsuru, Shuichi
PATENT ASSIGNEE(S): Oncotherapy Science, Inc., Japan; The University of Tokyo
SOURCE: PCT Int. Appl., 249pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2007013671	A2	20070201	WO 2006-JP315342	20060726
WO 2007013671	A3	20070830		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA				
EP 1907582	A2	20080409	EP 2006-782211	20060726
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				

JP 2009502116	T	20090129	JP 2008-504285		20060726
CN 101273144	A	20080924	CN 2006-80035233		20080324
PRIORITY APPLN. INFO.:			US 2005-703263P	P	20050727
			WO 2006-JP315342	W	20060726

AB In order to identify the mols. involved in esophageal carcinogenesis and those to be useful for diagnostic markers as well as targets for new drugs and immunotherapy, a cDNA microarray representing 32,256 genes was constructed to analyze the expression profiles of 19 esophageal squamous-cell carcinomas (ESCCS) purified by laser-capture microdissection. A detailed genome-wide database for sets of genes that are significantly up- or down-regulated in esophageal cancer is disclosed herein. These genes find use in the development of therapeutic drugs or immunotherapy as well as tumor markers. Addnl., genes associated with lymph-node metastasis and post-surgery recurrence are disclosed herein. Among the candidate mol. target genes, a Homo sapiens epithelial cell transforming sequence 2 oncogene (ECT2) and a cell division cycle 45, S. cerevisiae, homolog-like (CDC45L) are further characterized. Treatment of ESCC cells with small interfering RNAs (siRNAs) of ECT2 or CDC45L suppressed growth of the cancer cells. Thus, the data herein provide valuable information for identifying diagnostic systems and therapeutic target mols. for esophageal cancer. Furthermore, the present inventors have identified DKK1 as a potential biomarker for diagnosis of cancer such as lung and esophageal cancers as well as prediction of the poor prognosis of the patients with these diseases. DKK1 was specifically over-expressed in most lung and esophageal cancer tissues the present inventors examined, and was elevated in the sera of a large proportion of patients with these tumors. DKK1, combined with other tumor markers, could significantly improve the sensitivity of cancer diagnosis. Moreover, this mol. is also a likely candidate for development of therapeutic approaches such as antibody therapy.

L2 ANSWER 6 OF 14 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER:	2007:1213060 CAPLUS
DOCUMENT NUMBER:	147:462654
TITLE:	Steroid-regulated gene expression in the diagnosis of disease and selection of steroids for therapeutic use
INVENTOR(S):	Lal, Preeti; Rosenberg, Steven; Klinger, Tod
PATENT ASSIGNEE(S):	Expression Diagnostics, Inc., USA
SOURCE:	U.S. Pat. Appl. Publ., 25pp. CODEN: USXXCO
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	1
PATENT INFORMATION:	

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20070248978	A1	20071025	US 2007-784998	20070409
AU 2007309726	A1	20080502	AU 2007-309726	20070409
CA 2648580	A1	20080502	CA 2007-2648580	20070409
WO 2008051290	A2	20080502	WO 2007-US8909	20070409
WO 2008051290	A3	20081030		

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RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW,

GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

EP 2007909 A2 20081231 EP 2007-861283 20070409

R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, RS

IN 2008KN04491 A 20090313 IN 2008-KN4491 20081106

PRIORITY APPLN. INFO.: US 2006-790474P P 20060407

WO 2007-US8909 W 20070409

AB Genes that are regulated by steroid hormones are identified for use as markers in the diagnosis and monitoring of disease and in the selection of steroid hormone therapies. Specifically, genes that can be used as markers for the presence or absence of transplant rejection are identified.

L2 ANSWER 7 OF 14 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2007:936553 CAPLUS

DOCUMENT NUMBER: 148:446948

TITLE: A microarray analysis of retinal transcripts that are controlled by image contrast in mice

AUTHOR(S): Brand, Christine; Schaeffel, Frank; Feldkaemper, Marita Pauline

CORPORATE SOURCE: Section for Neurobiology of the Eye, University Eye Hospital Tuebingen, Tuebingen, Germany

SOURCE: Molecular Vision (2007), 13, 920-932
CODEN: MVEPFB; ISSN: 1090-0535
URL: http://www.molvis.org/molvis/v13/a98/v13a98-brand.pdf

PUBLISHER: Molecular Vision

DOCUMENT TYPE: Journal; (online computer file)

LANGUAGE: English

AB Purpose: The development of myopia is controlled by still largely unknown retinal signals. The aim of this study was to investigate the changes in retinal mRNA expression after different periods of visual deprivation in mice, while controlling for retinal illuminance. Methods: Each group consisted of three male C57BL/6 mice. Treatment periods were 30 min, 4 h, and 6+6 h. High spatial frequencies were filtered from the retinal image by frosted diffusers over one eye while the fellow eyes were covered by clear neutral d. (ND) filters that exhibited similar light attenuating properties (0.1 log units) as the diffusers. For the final 30 min of the resp. treatment period mice were individually placed in a clear Perspex cylinder that was positioned in the center of a rotating (60 degrees) large drum. The inside of the drum was covered with a 0.1 cyc/degree vertical square wave grating. This visual environment was chosen to standardize illuminances and contrasts seen by the mice. Labeled cRNA was prepared and hybridized to Affymetrix GeneChip Mouse Genome 430 2.0 arrays. Alterations in mRNA expression levels of candidate genes with potential biol. relevance were confirmed by semi-quant. real-time reverse transcription polymerase chain reaction (RT-PCR). Results: In all groups, Egr-1 mRNA expression was reduced in diffuser-treated eyes. Furthermore, the degradation of the spatial frequency spectrum also changed the cFos mRNA level, with reduced expression after 4 h of diffuser treatment. Other interesting candidates were Akt2, which was up-regulated after 30 min of deprivation and Mapk8i.p.3, a neuron specific JNK binding and scaffolding protein that was temporally regulated in the diffuser-treated eyes only. Conclusions: The microarray anal. demonstrated a pattern of differential transcriptional changes, even though differences in the retinal images were restricted to spatial features. The candidate genes may provide further insight into the biochem. short-term changes following retinal image degradation in mice. Because deprivation of spatial vision leads to increased eye growth and myopia in both animals and humans, it is believed some of the identified

genes play a role in myopia development.
REFERENCE COUNT: 74 THERE ARE 74 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 8 OF 14 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2006:1356154 CAPLUS
DOCUMENT NUMBER: 146:94691
TITLE: Gene map of human genes, single nucleotide polymorphisms, and haplotypes associated with longevity
INVENTOR(S): Belouchi, Abdelmajid; Raelson, John Verner; Bradley, Walter Edward; Paquin, Bruno; Nguyen-Huu, Quynh; Croteau, Pascal; Allard, Rene; Cousineau, Johanne; Paquin, Nouzha; Van Eerdewegh, Paul; Little, Randall David; Keith, Tim; Segal, Jonathan
PATENT ASSIGNEE(S): Genizon Biosciences, Inc., Can.
SOURCE: PCT Int. Appl., 219pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006138696	A2	20061228	WO 2006-US23724	20060619
WO 2006138696	A3	20070607		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA				
CA 2612389	A1	20061228	CA 2006-2612389	20060619
EP 1910569	A2	20080416	EP 2006-773488	20060619
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				

PRIORITY APPLN. INFO.: US 2005-691309P P 20050617
WO 2006-US23724 W 20060619

AB The present invention relates to the selection of a set of single nucleotide polymorphism (SNP) markers for use in genome-wide association studies based on linkage disequil. mapping. of a Quebec founder population. Genotyping was performed using the Perlegen Life Sciences ultra-high-throughput platform and allele discrimination through allele-specific hybridization. In total, 248,535 SNPs, spread over 3 microarrays, were genotyped. The raw data generated by the genome-wide association was analyzed by various means to identify candidate regions associated with longevity. A series of gene characterization steps was performed for each candidate region; any gene or EST mapping to the interval based on public map data or proprietary map data was considered as a candidate longevity gene. Candidate genes and regions were selected for sequencing in order to identify all polymorphisms., and once the major haplotypes were determined, appropriate genomic DNA samples were selected such that each major haplotype and haplotype subset were represented in at least two to four copies. 3741 SNPs were identified, and once the major haplotypes were determined, appropriate genomic DNA samples were selected such

that each major haplotype and haplotype subset were represented in at least two to four copies. The confirmation of putative assocns. was also performed in an independent general population patient sample. In particular, the invention relates to the fields of pharmacogenomics, diagnostics, patient therapy and the use of genetic haplotype information to predict an individual's longevity, their protection against age-related diseases, and/or their response to a particular drug or drugs.

L2 ANSWER 9 OF 14 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2006:1311584 CAPLUS
DOCUMENT NUMBER: 146:55471
TITLE: Gene expression markers for the identification, assessment, and treatment, and responsiveness of cancer using proteasome inhibition or glucocorticoid therapy
INVENTOR(S): Bryant, Barbara M.; Damokosh, Andrew I.; Mulligan, George
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 152pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006133420	A2	20061214	WO 2006-US22515	20060608
WO 2006133420	A3	20080117		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA				
AU 2006254834	A1	20061214	AU 2006-254834	20060608
CA 2611728	A1	20061214	CA 2006-2611728	20060608
US 20060281122	A1	20061214	US 2006-449195	20060608
EP 1899486	A2	20080319	EP 2006-784710	20060608
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU				
JP 2008544223	T	20081204	JP 2008-515977	20060608
MX 2007015416	A	20080219	MX 2007-15416	20071206
PRIORITY APPLN. INFO.:			US 2005-688634P	P 20050608
			WO 2006-US22515	W 20060608

AB The present invention is directed to the identification of predictive markers that can be used to determine whether patients with cancer are clin. responsive or non-responsive to a therapeutic regimen prior to treatment. In particular, the present invention is directed to the use of certain individual and/or combinations of predictive markers, wherein the expression of the predictive markers correlates with responsiveness or non-responsiveness to a proteasome inhibition and/or a glucocorticoid therapeutic regimen. A multicenter, open-label, randomized study was conducted comprising 627 enrolled patients with relapsed or refractory multiple myeloma treated with either bortezomib (Velcade) or

dexamethasone (Decodron). Differentially expressed markers on Affymetrix U133 microarrays (A and B) were identified by using a combination of marker ranking algorithms, supervised learning, and feature selection algorithms. The expression levels of individual predictive markers, and/or predictive markers comprising a marker set, are correlated with a pos. or neg. response to therapy or a long time until disease progression.

L2 ANSWER 10 OF 14 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2006:1157631 CAPLUS
DOCUMENT NUMBER: 145:483673
TITLE: Novel methods and devices for evaluating poisons
INVENTOR(S): Ching, Edwin P.; Johnson, Dale E.; Sudarsanam, Sucha
PATENT ASSIGNEE(S): Emiliem, USA
SOURCE: PCT Int. Appl., 132pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006116622	A2	20061102	WO 2006-US16067	20060426
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
US 20060253262	A1	20061109	US 2006-380388	20060426
EP 1880332	A2	20080123	EP 2006-751675	20060426
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JP 2008541696	T	20081127	JP 2008-509135	20060426
IN 2007DN08071	A	20080704	IN 2007-DN8071	20071018
PRIORITY APPLN. INFO.:			US 2005-675741P	P 20050427
			US 2006-778133P	P 20060301
			WO 2006-US16067	W 20060426

AB Methods and devices useful for evaluating poisons or other chemical entities, and for using such methods to forecast unfavorable drug effects. The present invention provides lists of biomarkers for anal., either directly or indirectly, which affect the toxicity pathways. These may be evaluated at many levels, including genetic, genotyping, evaluation of combination pairing of diploid alleles or haplotypes, RNA expression, protein expression, functional activity, posttranslational anal. or evaluation, etc. Thus, the biomarkers refer to the corresponding genetic information, RNA, protein, or other structural embodiments thereof. And the means to use these biomarkers, e.g., to evaluate status of toxicity pathways, to evaluate individual risk or susceptibility to various toxic pathways from exposure or therapeutic intervention, to generate test systems for drug development, are all provided by identifying critical and significant contributors to the pathway progression. The present invention is directed to accelerating the speed of development and reducing the resource investment necessary to determine these features for directing use of such substances or treatments to appropriate biol. contexts.

L2 ANSWER 11 OF 14 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2005:1103600 CAPLUS
DOCUMENT NUMBER: 143:360117
TITLE: Adiponectin expression inducer and utilization of the same
INVENTOR(S): Kadowaki, Takashi; Yamauchi, Toshimasa; Kitajima, Shoko; Ito, Yusuke
PATENT ASSIGNEE(S): Toudai Tlo, Ltd., Japan; Mitsubishi Pharma Corporation; Nissan Chemical Industries, Ltd.
SOURCE: PCT Int. Appl., 50 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005094866	A1	20051013	WO 2005-JP6357	20050331
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1757302	A1	20070228	EP 2005-727594	20050331
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR				
US 20070203061	A1	20070830	US 2007-594969	20070314
PRIORITY APPLN. INFO.:			US 2004-557708P	P 20040331
			WO 2005-JP6357	W 20050331

AB It is intended to provide an adiponectin expression inducer, a remedy for obesity and diseases relating thereto, for example, cardiovascular diseases or metabolic diseases by using the same, and a method of searching for an adiponectin expression inducer. It is indicated that KLF9, which is capable of binding to a 32 bp fragment of from -188- to -157-positions starting with the expression initiator of adiponectin, can enhance the adiponectin promoter activity. Therefore, it is suggested that KLF9 supplemental therapy with the use of KLF9 as an adiponectin expression inducer might contribute to the prevention and treatment for obesity or diseases relating thereto, for example, metabolic diseases such as insulin resistance diabetes and type 2 diabetes and cardiovascular diseases.
REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 12 OF 14 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2005:216606 CAPLUS
DOCUMENT NUMBER: 142:292452
TITLE: Compns. and methods for treating and diagnosing chronic visceral hypersensitivity and irritable bowel syndrome, based on differential gene or protein expression
INVENTOR(S): Pasricha, Pankaj; Shenoy, Mohan; Winston, John
PATENT ASSIGNEE(S): Cytokine Pharmasciences, Inc., USA
SOURCE: PCT Int. Appl., 181 pp.

CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005020902	A2	20050310	WO 2004-US27356	20040823
WO 2005020902	A3	20060727		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 20050130189	A1	20050616	US 2004-923035	20040823

PRIORITY APPLN. INFO.: US 2003-496716P P 20030821

AB Compns. and methods for diagnosing and treating chronic visceral hypersensitivity (CVH) and CVH-associated disorders, such as irritable bowel syndrome, are disclosed. Genes differentially expressed in CVH tissues relative to normal tissues are identified. The genes and the gene products (i.e., the transcribed polynucleotides and polypeptides encoded by the genes) can be used as markers of CVH. The genes and the gene products can also be used to screen agents that modulate the gene expression or the activities of the gene products. The examples discuss the effects of acetic acid sensitization and CNI1493 treatment on the colon and S1 dorsal root ganglia in a rat model of visceral hypersensitivity. Gene expression profiles associated with these treatments are presented, and rat CVH-related genes and polypeptides are identified.

L2 ANSWER 13 OF 14 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2005:66610 CAPLUS
DOCUMENT NUMBER: 142:368954
TITLE: Identification of novel TCDD-regulated genes by microarray analysis
AUTHOR(S): Hanlon, Paul R.; Zheng, Wenchao; Ko, Alex Y.; Jefcoate, Colin R.
CORPORATE SOURCE: Molecular and Environmental Toxicology Center, University of Wisconsin-Madison, WI, 53706, USA
SOURCE: Toxicology and Applied Pharmacology (2005), 202(3), 215-228
CODEN: TXAPA9; ISSN: 0041-008X
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English

AB TCDD exposure of multipotential C3H10T1/2 fibroblasts for 72 h altered the expression of over 1000 genes, including coordinated changes across large functionally similar gene clusters. TCDD coordinately induced 23 cell cycle-related genes similar to epidermal growth factor (EGF)-induced levels but without any affect on the major mitogenic signaling pathway (extracellular signal-regulated kinase, ERK). TCDD treatment also decreased glycolytic and ribosomal clusters. Most of these TCDD-induced changes were attenuated by the presence of EGF or an adipogenic stimulus, each added during the final 24 h. TCDD prevented 10% of EGF-induced gene responses and 40% of adipogenic responses. Over 100

other genes responded to TCDD during adipogenesis. This group of responses included complete suppression of three proliferins and stimulations of several cytokine receptors. Despite these varied secondary effects of TCDD, direct AhR activation measured by integrated AhR-responsive luciferase reporters was similar under quiescent, EGF-stimulated or adipogenic conditions. Only 23 genes were similarly induced by TCDD regardless of conditions and 10 were suppressed. These 23 genes include: 4 genes previously recognized to contain AhR response elements (cytochrome P 450 (CYP) 1B1, CYP1A1, NAD(P)H quinone reductase 1 (NQO1), and aldehyde dehydrogenase 3A1); two novel oxidative genes (alc. dehydrogenase 3 and superoxide dismutase 3); and glypican 1, a plasma membrane proteoglycan that affects cell signaling. Further expts. demonstrated that TCDD maximally induced NQO1, glypican 1 and alc. dehydrogenase 3 by 6 h. Glypican 1 activates the actions of many growth factors and therefore may contribute to secondary effects on gene expression.

REFERENCE COUNT: 61 THERE ARE 61 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 14 OF 14 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2005377822 EMBASE

TITLE: Null mutation of Kruippel-like factor9/basic transcription element binding protein-1 alters peri-implantation uterine development in mice.

AUTHOR: Velarde, Michael C.; Van Geng; Simmen, Frank A.; Simmen, Rosalia C. M. (correspondence)

CORPORATE SOURCE: Department of Physiology and Biophysics, University of Arkansas for Medical Sciences, Little Rock, AR 72202, United States. simmenrosalia@uams.edu

AUTHOR: Eason, Renea R.; Simmen, Frank A.; Simmen, Rosalia C. M. (correspondence)

CORPORATE SOURCE: University of Arkansas for Medical Sciences, Arkansas Children's Nutrition Center, 1120 Marshall St., Little Rock, AR 72202, United States. simmenrosalia@uams.edu

SOURCE: Biology of Reproduction, (Sep 2005) Vol. 73, No. 3, pp. 472-481.
Refs: 44
ISSN: 0006-3363 CODEN: BIREBV

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 021 Developmental Biology and Teratology

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 22 Sep 2005
Last Updated on STN: 22 Sep 2005

AB Female mice null for the basic transcription element binding protein-1 (Bteb1) gene have reduced numbers of implanting embryos. We hypothesized that the implantation defect, resulting in subfertility, is a consequence of developmental asynchrony between the embryo and uterine endometrium at peri-implantation. To address this, endometrium from wild-type (WT) and Bteb1((-/-)) females at 0.5 to 5.5 days postcoitum (dpc) were evaluated for proliferation (BrdU labeling), apoptosis (TUNEL), and steroid hormone receptor expression (immunohistochemistry). Loss of BTEB1 did not affect serum estrogen (E) and progesterone (P) levels. In stroma (ST), the numbers of progesterone receptor (PGR) and HomeoboxA10 (HOXA10)-expressing cells were lower (3.5 and 4.5 dpc), while those of estrogen receptor-alpha (ESR1) were higher (3.5 dpc), with Bteb1 ablation. The peak of proliferation in luminal epithelium (LE), glandular epithelium (CE), and ST was delayed, while the apoptotic index in all cell types was increased (2.5 dpc) in Bteb1((-/-)) relative to WT mice. The numbers of PGR-positive ST cells was negatively correlated with LE proliferation in

WT mice; this correlation was lost in Btebl((-/-)) mice and was not observed before 2.5 dpc for both genotypes. Proliferation and apoptosis in all endometrial compartments, as well as the numbers of PGR-, HOXA10-, and ESR1-expressing ST cells, were lower in Btebl((-/-)) relative to WT mice after ovariectomy and E + P treatment. Results suggest that BTEB1, by regulating ST PGR expression and transactivation, participates in the paracrine control of LE proliferation by PGR and thus is important for establishment of a receptive uterus critical for successful implantation. .COPYRGT. 2005 by the Society for the Study of Reproduction, Inc.

=> Log off h
SESSION WILL BE HELD FOR 120 MINUTES
STN INTERNATIONAL SESSION SUSPENDED AT 14:55:41 ON 28 APR 2009

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:SSPTAEGS1646

PASSWORD:
* * * * * * RECONNECTED TO STN INTERNATIONAL * * * * * *
SESSION RESUMED IN FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE'
AT 14:57:44 ON 28 APR 2009
FILE 'MEDLINE' ENTERED AT 14:57:44 ON 28 APR 2009
FILE 'BIOSIS' ENTERED AT 14:57:44 ON 28 APR 2009
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FILE 'CAPLUS' ENTERED AT 14:57:44 ON 28 APR 2009
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FILE 'EMBASE' ENTERED AT 14:57:44 ON 28 APR 2009
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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	49.78	50.00
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-8.20	-8.20

=> D Hist

(FILE 'HOME' ENTERED AT 14:53:55 ON 28 APR 2009)

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 14:54:20 ON 28 APR 2009
L1 20 S (KRUPPEL-LIKE FACTOR 9) AND TREATMENT
L2 14 DUP REM L1 (6 DUPLICATES REMOVED)

=> S (KRUPPEL-LIKE FACTOR 9) (S) (adiponectin OR Acrp30) AND pd<=20050331
2 FILES SEARCHED...
L3 0 (KRUPPEL-LIKE FACTOR 9) (S) (ADIPONECTIN OR ACRP30) AND PD<=20050331

=> S adiponectin (S) induc? (S) factor AND pd<=20050331
2 FILES SEARCHED...
L4 95 ADIPONECTIN (S) INDUC? (S) FACTOR AND PD<=20050331

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=> Dup Rem L4
PROCESSING COMPLETED FOR L4
L5            54 DUP REM L4 (41 DUPLICATES REMOVED)
                 ANSWERS '1-21' FROM FILE MEDLINE
                 ANSWERS '22-23' FROM FILE BIOSIS
                 ANSWERS '24-53' FROM FILE CAPLUS
                 ANSWER '54' FROM FILE EMBASE

=> D Ti L5 1-54

L5   ANSWER 1 OF 54     MEDLINE on STN                  DUPLICATE 1
TI   Adiponectin inhibits cell proliferation by interacting with several growth
     factors in an oligomerization-dependent manner.

L5   ANSWER 2 OF 54     MEDLINE on STN                  DUPLICATE 2
TI   Leptin and adiponectin stimulate the release of proinflammatory cytokines
     and prostaglandins from human placenta and maternal adipose tissue via
     nuclear factor-kappaB, peroxisomal proliferator-activated receptor-gamma
     and extracellularly regulated kinase 1/2.

L5   ANSWER 3 OF 54     MEDLINE on STN                  DUPLICATE 3
TI   Autocrine action of adiponectin on human fat cells prevents the
     release of insulin resistance-inducing factors.

L5   ANSWER 4 OF 54     MEDLINE on STN                  DUPLICATE 4
TI   Pathophysiology of metabolic syndrome X and its links to the perinatal
     period.

L5   ANSWER 5 OF 54     MEDLINE on STN                  DUPLICATE 5
TI   Changes in insulin sensitivity induced by short-term growth
     hormone (GH) and insulin-like growth factor I (IGF-I) treatment
     in GH-deficient adults are not associated with changes in
     adiponectin levels.

L5   ANSWER 6 OF 54     MEDLINE on STN                  DUPLICATE 6
TI   Fatty acids and expression of adipokines.

L5   ANSWER 7 OF 54     MEDLINE on STN                  DUPLICATE 7
TI   Globular adiponectin decreases leptin-induced tumor
     necrosis factor-alpha expression by murine macrophages:
     involvement of cAMP-PKA and MAPK pathways.

L5   ANSWER 8 OF 54     MEDLINE on STN                  DUPLICATE 8
TI   Obesity as the core of the metabolic syndrome and the management of
     coronary heart disease.

L5   ANSWER 9 OF 54     MEDLINE on STN                  DUPLICATE 9
TI   Adiponectin, hepatocellular dysfunction and insulin sensitivity.

L5   ANSWER 10 OF 54     MEDLINE on STN                 DUPLICATE 10
TI   Adiponectin protects LPS-induced liver injury through modulation of
     TNF-alpha in KK-Ay obese mice.

L5   ANSWER 11 OF 54     MEDLINE on STN                 DUPLICATE 11
TI   Diabetes, lipids, and adipocyte secretagogues.

L5   ANSWER 12 OF 54     MEDLINE on STN                 DUPLICATE 12
TI   Enhanced carbon tetrachloride-induced liver fibrosis in mice lacking
     adiponectin.

L5   ANSWER 13 OF 54     MEDLINE on STN                 DUPLICATE 13
TI   Induction of adiponectin, a fat-derived antidiabetic
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and antiatherogenic factor, by nuclear receptors.

L5 ANSWER 14 OF 54 MEDLINE on STN DUPLICATE 14
TI Obesity, adiponectin and vascular inflammatory disease.

L5 ANSWER 15 OF 54 MEDLINE on STN DUPLICATE 15
TI Adipocyte-specific gene expression and adipogenic steatosis in the mouse liver due to peroxisome proliferator-activated receptor gamma1 (PPARgamma1) overexpression.

L5 ANSWER 16 OF 54 MEDLINE on STN DUPLICATE 16
TI Role of PPARs in the regulation of obesity-related insulin sensitivity and inflammation.

L5 ANSWER 17 OF 54 MEDLINE on STN DUPLICATE 17
TI Role of adiponectin in preventing vascular stenosis. The missing link of adipo-vascular axis.

L5 ANSWER 18 OF 54 MEDLINE on STN DUPLICATE 18
TI Adipocyte-derived plasma protein adiponectin acts as a platelet-derived growth factor-BB-binding protein and regulates growth factor-induced common postreceptor signal in vascular smooth muscle cell.

L5 ANSWER 19 OF 54 MEDLINE on STN DUPLICATE 19
TI Adiponectin, a new member of the family of soluble defense collagens, negatively regulates the growth of myelomonocytic progenitors and the functions of macrophages.

L5 ANSWER 20 OF 54 MEDLINE on STN DUPLICATE 20
TI Adiponectin, an adipocyte-derived plasma protein, inhibits endothelial NF-kappaB signaling through a cAMP-dependent pathway.

L5 ANSWER 21 OF 54 MEDLINE on STN
TI Adipose tissue as an endocrine organ.

L5 ANSWER 22 OF 54 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
TI Autocrine action of adiponectin on human fat cells prevents the release of insulin resistance-inducing factors.

L5 ANSWER 23 OF 54 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
TI AMP-activated protein kinase (AMPK) activator, AICAR and adiponectin inhibit vascular endothelial growth factor (VEGF) induced vascular cell adhesion molecule (VCAM)-1 expression in human aortic endothelial cells (HAEC).

L5 ANSWER 24 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Adiponectin inhibits Toll-like receptor family-induced signaling

L5 ANSWER 25 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Roles of adiponectin in the regulation of metabolism

L5 ANSWER 26 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Pathogenesis and molecular mechanism of metabolic syndrome

L5 ANSWER 27 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Adiponectin induces TNF-α and IL-6 in macrophages and promotes tolerance to itself and other pro-inflammatory stimuli

L5 ANSWER 28 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN

TI Adiponectin inhibits LPS-induced NF-κB activation and IL-6 production and increases PPARγ2 expression in adipocytes

L5 ANSWER 29 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Adiponectin increases bone mass by suppressing osteoclast and activating osteoblast

L5 ANSWER 30 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Hyperglycemia- and hyperinsulinemia-induced alteration of adiponectin receptor expression and adiponectin effects in L6 myoblasts

L5 ANSWER 31 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Metabolic syndrome. Pathological state of visceral fat accumulation syndrome and arteriosclerosis

L5 ANSWER 32 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI PPARs and glucose/lipid metabolism

L5 ANSWER 33 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Adipokines and insulin resistance

L5 ANSWER 34 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Adipocyte molecular and cellular biology in the metabolic syndrome

L5 ANSWER 35 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Cancer and adiponectin

L5 ANSWER 36 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Metabolic syndrome and circulatory diseases

L5 ANSWER 37 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Adiponectin suppresses proliferation and proinflammatory cytokine production in THP-1 monocytes, and induces caspase 3/7 activity

L5 ANSWER 38 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Adiponectin

L5 ANSWER 39 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Phosphorylation of C/EBPβ at a consensus extracellular signal-regulated kinase/glycogen synthase kinase 3 site is required for the induction of adiponectin gene expression during the differentiation of mouse fibroblasts into adipocytes

L5 ANSWER 40 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Induction of adiponectin in skeletal muscle by inflammatory cytokines: In vivo and in vitro studies

L5 ANSWER 41 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI CCAAT/enhancer binding protein and nuclear factor-Y regulate adiponectin gene expression in adipose tissue

L5 ANSWER 42 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Metabolic syndrome and mechanism of action of adipocytokines

L5 ANSWER 43 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Adiponectin receptors

L5 ANSWER 44 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI NASH and adipocytokine

L5 ANSWER 45 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Adiponectin counteracts cytokine- and fatty acid-induced apoptosis in the

pancreatic beta-cell line INS-1

L5 ANSWER 46 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Sustained peripheral expression of transgene adiponectin offsets the development of diet-induced obesity in rats

L5 ANSWER 47 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Plasma acylation-stimulating protein, adiponectin, leptin, and ghrelin before and after weight loss induced by gastric bypass surgery in morbidly obese subjects

L5 ANSWER 48 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Insulin resistance and role of renin-angiotensin system

L5 ANSWER 49 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI The multivariate functions of adipose tissue and the metabolic syndrome: the effects of soybean protein on fat distribution and adipocytokines

L5 ANSWER 50 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Human cDNA sequences and their encoded proteins and diagnostic and therapeutic uses

L5 ANSWER 51 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Diet-induced insulin resistance in mice lacking adiponectin/ACRP30

L5 ANSWER 52 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI Adiponectin and 5'-AMP-activated protein kinase

L5 ANSWER 53 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
TI FFA as adipocytokines in atherosclerosis

L5 ANSWER 54 OF 54 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI Role of PPARs in the regulation of obesity-related insulin sensitivity and inflammation.

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L1 20 S (KRUPPEL-LIKE FACTOR 9) AND TREATMENT
L2 14 DUP REM L1 (6 DUPLICATES REMOVED)
L3 0 S (KRUPPEL-LIKE FACTOR 9) (S) (ADIPONECTIN OR ACRP30) AND PD<=2
L4 95 S ADIPONECTIN (S) INDUC? (S) FACTOR AND PD<=20050331
L5 54 DUP REM L4 (41 DUPLICATES REMOVED)

=> D Ibib abs L5 7-9, 11, 13, 14, 31,38,43,48

L5 ANSWER 7 OF 54 MEDLINE on STN DUPLICATE 7
ACCESSION NUMBER: 2006116638 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16438946
TITLE: Globular adiponectin decreases leptin-induced tumor necrosis factor-alpha expression by murine macrophages: involvement of cAMP-PKA and MAPK pathways.
AUTHOR: Zhao Ting; Hou Mengjun; Xia Min; Wang Qing; Zhu Huilian; Xiao Yongmei; Tang Zhihong; Ma Jing; Ling Wenhua
CORPORATE SOURCE: Department of Nutrition, School of Public Health, Zhongshan University (Northern Campus), Guangzhou, Guangdong Province, PR China.
SOURCE: Cellular immunology, (2005 Nov) Vol. 238, No. 1, pp. 19-30. Electronic Publication: 2006-01-24. Journal code: 1246405. ISSN: 0008-8749.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200604
ENTRY DATE: Entered STN: 1 Mar 2006
Last Updated on STN: 14 Apr 2006
Entered Medline: 13 Apr 2006

AB Several lines of evidence have supported a link between obesity and inflammation. The present study investigated the capacity of leptin and globular adiponectin to affect tumor necrosis factor alpha (TNF-alpha) production in murine peritoneal macrophages. Leptin stimulated TNF-alpha production at mRNA as well as protein levels in a dose- and time-dependent manner. Intracellular cAMP concentration was increased and protein kinase A (PKA) was activated with the treatment of leptin, subsequently downstream MAPK signal proteins, ERK1/2 and p38, were phosphorylated. Specific inhibitors for the signal proteins, Rp cAMPS, H89, PD98059, and U0126, or SB203580, suppressed the signaling pathway and TNF-alpha expression. Although gAd partially increased cAMP concentration and PKA activity, it directly reduced leptin-induced ERK1/2 and p38 MAPK phosphorylation thus inhibiting TNF-alpha production. In conclusion, leptin promotes inflammation by stimulating TNF-alpha production, which is mediated by cAMP-PKA-ERK1/2 and p38 MAPK pathways. gAd inhibited leptin-induced TNF-alpha production through suppressing phosphorylation of ERK1/2 and p38 pathways.

L5 ANSWER 8 OF 54 MEDLINE on STN DUPLICATE 8
ACCESSION NUMBER: 2004133651 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15025838
TITLE: Obesity as the core of the metabolic syndrome and the management of coronary heart disease.
AUTHOR: Shirai Kohji
CORPORATE SOURCE: Center for Diabetes, Endocrinology and Metabolism, Sakura Hospital, Toho University, Shimoshizu, Japan.. kshira@kb3.so-net.ne.jp
SOURCE: Current medical research and opinion, (2004 Mar) Vol. 20, No. 3, pp. 295-304. Ref: 111
Journal code: 0351014. ISSN: 0300-7995.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200406
ENTRY DATE: Entered STN: 18 Mar 2004
Last Updated on STN: 11 Jun 2004
Entered Medline: 10 Jun 2004

AB The global burden of coronary heart disease (CHD) has led to the introduction of international guidelines to minimize the morbidity and mortality that result from this condition. These guidelines recognize the contribution of multiple risk factors to the development of CHD and advocate a multifaceted approach to treatment. Obesity, particularly visceral adiposity, contributes to the clustering of many other risk factors, such as hypertension, insulin resistance/type 2 diabetes and dyslipidemia, within individual patients. The molecular mechanisms underlying the metabolic abnormalities induced by visceral adiposity have yet to be fully elucidated; however, adipocytokines such as adiponectin, tumor necrosis factor-alpha and resistin seem to play an important role in this process. Obesity is a major modifiable CHD risk factor, and the benefits of weight loss are numerous, leading to improvements in several co-morbidities. Guidelines advocate lifestyle changes to correct excess bodyweight and improve the CHD risk factor profile. In addition, pharmacologic therapy is recommended for the management of other risk factors, such as hypertension and dyslipidemia, which may not be adequately controlled by lifestyle changes alone. Lowering low-density lipoprotein cholesterol (LDL-C) levels is the primary target for drug therapy for CHD prevention, and statins are first-line lipid-modifying therapy.The introduction of more efficacious statins with favorable effects on the lipid profile will optimize the control of dyslipidemia. Combining these new treatments with lifestyle changes and drug therapies for managing other CHD risk factors, as part of a multifaceted approach to treatment, will have benefits for CHD prevention.

L5 ANSWER 9 OF 54 MEDLINE on STN DUPLICATE 9
ACCESSION NUMBER: 2004070988 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14725689
TITLE: Adiponectin, hepatocellular dysfunction and insulin sensitivity.
AUTHOR: Lopez-Bermejo Abel; Botas Patricia; Funahashi Tohru; Delgado Elias; Kihara Shinji; Ricart Wifredo; Fernandez-Real Jose Manuel
CORPORATE SOURCE: Unit of Diabetes, Endocrinology and Nutrition, University Hospital of Girona Dr Josep Trueta, Girona, Spain.. uden.alopez@htrueta.scs.es
SOURCE: Clinical endocrinology, (2004 Feb) Vol. 60, No. 2, pp. 256-63.

Journal code: 0346653. ISSN: 0300-0664.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200403
ENTRY DATE: Entered STN: 13 Feb 2004
Last Updated on STN: 26 Mar 2004
Entered Medline: 25 Mar 2004

AB OBJECTIVE: Insulin resistance plays a major aetiological role in the development of fatty liver disease. Because adiponectin is a hepatic insulin sensitizer and also an inhibitor of tumour necrosis factor, a cytokine known to induce insulin resistance and liver damage, we wished to study whether low circulating adiponectin would be associated with higher serum concentrations of liver enzymes in healthy subjects. DESIGN: Cross-sectional, population-based study dealing with diabetes prevalence in northern Spain. PATIENTS: Two hundred and fifty-seven apparently healthy Caucasian subjects consecutively enrolled in the study. MEASUREMENTS: Adiponectin serum levels were measured by enzyme-linked immunosorbent assay (ELISA), liver function tests (LFTs) by colourimetry and insulin resistance by the homeostasis model of assessment (HOMA value). RESULTS: Adiponectin levels were negatively correlated with alanine aminotransferase (ALT) and gamma-glutamyltranspeptidase (GGT), before and after adjustment for sex, age, body mass index (BMI) and insulin resistance (ALT; r = -0.32, P < 0.001; adjusted: r = -0.13, P = 0.033; GGT; r = -0.31, P < 0.001; adjusted: r = -0.16, P = 0.011). Additionally, adiponectin correlated with alkaline phosphate (ALKP) only after adjusting for the same confounding variables (r =-0.10, P = 0.098; adjusted: r = -0.14, P = 0.031). A general linear model, adjusting for age, sex and BMI, was constructed to predict the decrease in circulating adiponectin for each LFT value (i.e. ALT, GGT and ALKP) above the median. Beyond one LFT value above the median, serum adiponectin decreased by -0.97 mg/l (95% CI -1.46 to -0.48). In multiple regression analysis, sex, BMI and adiponectin, but not insulin resistance, predicted serum concentrations of both ALT and GGT, explaining 19% and 14% of their variance, respectively. Age, BMI and adiponectin, but not sex or insulin resistance, explained 20% of ALKP variance. CONCLUSIONS: Adiponectin levels are associated in healthy humans with plasma concentrations of various liver function tests. The contributions of adiponectin to maintaining liver integrity through the regulation of both insulin sensitivity and/or the inflammatory response merit further studies.

L5 ANSWER 11 OF 54 MEDLINE on STN DUPLICATE 11
ACCESSION NUMBER: 2004214490 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15052336
TITLE: Diabetes, lipids, and adipocyte secretagogues.
AUTHOR: Faraj May; Lu Hui Ling; Cianflone Katherine
CORPORATE SOURCE: Mike Rosenbloom Laboratory for Cardiovascular Research, McGill University Health Centre, Royal Victoria Hospital, Montreal, QC, Canada.
SOURCE: Biochemistry and cell biology = Biochimie et biologie cellulaire, (2004 Feb) Vol. 82, No. 1, pp. 170-90. Ref: 283
Journal code: 8606068. ISSN: 0829-8211.
PUB. COUNTRY: Canada
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 200411
ENTRY DATE: Entered STN: 29 Apr 2004
Last Updated on STN: 3 Nov 2004
Entered Medline: 2 Nov 2004

AB That obesity is associated with insulin resistance and type II diabetes mellitus is well accepted. Overloading of white adipose tissue beyond its storage capacity leads to lipid disorders in non-adipose tissues, namely skeletal and cardiac muscles, pancreas, and liver, effects that are often mediated through increased non-esterified fatty acid fluxes. This in turn leads to a tissue-specific disordered insulin response and increased lipid deposition and lipotoxicity, coupled to abnormal plasma metabolic and (or) lipoprotein profiles. Thus, the importance of functional adipocytes is crucial, as highlighted by the disorders seen in both "too much" (obesity) and "too little" (lipodystrophy) white adipose tissue. However, beyond its capacity for fat storage, white adipose tissue is now well recognised as an endocrine tissue producing multiple hormones whose plasma levels are altered in obese, insulin-resistant, and diabetic subjects. The consequence of these hormonal alterations with respect to both glucose and lipid metabolism in insulin target tissues is just beginning to be understood. The present review will focus on a number of these hormones: acylation-stimulating protein, leptin, adiponectin, tumour necrosis factor alpha, interleukin-6, and resistin, defining their changes induced in obesity and diabetes mellitus and highlighting their functional properties that may protect or worsen lipid metabolism.

L5 ANSWER 13 OF 54 MEDLINE on STN DUPLICATE 13
ACCESSION NUMBER: 2003349465 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12829629
TITLE: Induction of adiponectin, a fat-derived antidiabetic and antiatherogenic factor, by nuclear receptors.
AUTHOR: Iwaki Masanori; Matsuda Morihiro; Maeda Norikazu; Funahashi Tohru; Matsuzawa Yuji; Makishima Makoto; Shimomura Iichiro
CORPORATE SOURCE: Department of Medicine and Pathophysiology, Graduate School of Frontier Biosciences, Graduate School of Medicine, Osaka University, Suita, Japan.
SOURCE: Diabetes, (2003 Jul) Vol. 52, No. 7, pp. 1655-63.
Journal code: 0372763. ISSN: 0012-1797.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200308
ENTRY DATE: Entered STN: 29 Jul 2003
Last Updated on STN: 20 Aug 2003
Entered Medline: 19 Aug 2003

AB Adiponectin is a fat-derived hormone with antidiabetic and antiatherogenic properties. Hypoadiponectinemia seen in obesity is associated with insulin-resistant diabetes and atherosclerosis. Thiazolidinediones, peroxisome proliferator-activated receptor-gamma (PPAR-gamma) agonists, have been shown to increase plasma adiponectin levels by the transcriptional induction in adipose tissues. However, the precise mechanism of such action is unknown. In this study, we have identified a functional PPAR-responsive element (PPRE) in human adiponectin promoter. PPAR-gamma/retinoid X receptor (RXR) heterodimer directly bound to the PPRE and increased the promoter activity in cells. In adipocytes, point mutation of the PPRE markedly reduced the basal transcriptional activity and completely blocked thiazolidinedione-induced transactivation of adiponectin promoter. We have also identified a responsive element of another orphan nuclear receptor, liver receptor homolog-1 (LRH-1), in

adiponectin promoter. LRH-1 was expressed in 3T3-L1 cells and rat adipocytes. LRH-1 bound specifically to the identified responsive element (LRH-RE). LRH-1 augmented PPAR-gamma-induced transactivation of adiponectin promoter, and point mutation of the LRH-RE significantly decreased the basal and thiazolidinedione-induced activities of adiponectin promoter. Our results indicate that PPAR-gamma and LRH-1 play significant roles in the transcriptional activation of adiponectin gene via the PPRE and the LRH-RE in its promoter.

L5 ANSWER 14 OF 54 MEDLINE on STN DUPLICATE 14
ACCESSION NUMBER: 2003543810 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14624132
TITLE: Obesity, adiponectin and vascular inflammatory disease.
AUTHOR: Ouchi Noriyuki; Kihara Shinji; Funahashi Tohru; Matsuzawa Yuji; Walsh Kenneth
CORPORATE SOURCE: Department of Internal Medicine and Molecular Science, Graduate School of Medicine, Osaka University, Suita, Osaka, Japan.. ouchi@imed2.med.osaka-u.ac.jp
SOURCE: Current opinion in lipidology, (2003 Dec) Vol. 14, No. 6, pp. 561-6. Ref: 49
Journal code: 9010000. ISSN: 0957-9672.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200502
ENTRY DATE: Entered STN: 19 Nov 2003
Last Updated on STN: 14 Jul 2004
Entered Medline: 15 Feb 2005
AB PURPOSE OF REVIEW: Obesity is the most common risk factor for cardiovascular diseases in industrial countries. It is now clear that adipose tissue secretes various bioactive substances, conceptualized as adipocytokines, and that dysregulation of adipocytokines directly contributes to obesity-related diseases. Chronic inflammatory processes contribute to the development of atherosclerosis. In this review, the authors focus on the relationship between adiponectin, a recently discovered anti-atherogenic adipocytokine, and vascular inflammation. RECENT FINDINGS: Plasma concentrations of adiponectin, an adipocyte-specific protein, are reduced in obese subjects and in patients with type 2 diabetes and coronary artery disease. Adiponectin inhibits the expression of tumor necrosis factor-alpha-induced endothelial adhesion molecules, macrophage-to-foam cell transformation, tumor necrosis factor-alpha expression in macrophages and adipose tissues, and smooth muscle cell proliferation. In addition, adenovirus-expressed adiponectin reduces atherosclerotic lesions in a mouse model of atherosclerosis, and adiponectin-deficient mice exhibit an excessive vascular remodeling response to injury. Clinically, hypoadiponectinemia is closely associated with increased levels of inflammatory markers such as C-reactive protein and interleukin-6. SUMMARY: Adiponectin acts as an anti-inflammatory and anti-atherogenic plasma protein. Adiponectin is an endogenous biologically relevant modulator of vascular remodeling linking obesity and vascular disease.

L5 ANSWER 31 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2005:273732 CAPLUS
DOCUMENT NUMBER: 142:352725
TITLE: Metabolic syndrome. Pathological state of visceral fat accumulation syndrome and arteriosclerosis
AUTHOR(S): Funahashi, Toru
CORPORATE SOURCE: Grad. Sch. Med., Osaka Univ., Japan

SOURCE: Nippon Naika Gakkai Zasshi (2005), 94(3), 405-410
CODEN: NNGAAS; ISSN: 0021-5384
PUBLISHER: Nippon Naika Gakkai
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese
AB A review on pathophysiol. of metabolic syndrome (visceral fat syndrome) as multiple risk factor syndrome in atherosclerosis, role of adiponectin in visceral fat accumulation-induced metabolic syndrome, and management of metabolic syndrome.

L5 ANSWER 38 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2005:197212 CAPLUS
DOCUMENT NUMBER: 143:41511
TITLE: Adiponectin
AUTHOR(S): Maeda, Norikazu; Funahashi, Tohru; Shimomura, Iichiro
CORPORATE SOURCE: Graduate School of Medicine, Osaka University, Japan
SOURCE: Molecular Medicine (Tokyo, Japan) (2005), 42(1), 11-21
CODEN: MOLMEL; ISSN: 0918-6557
PUBLISHER: Nakayama Shoten
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese
AB A review on roles of adipocyte-derived adiponectin in metabolic syndrome. The topics discussed are (1) adipocyte-derived adiponectin; (2) structure of adiponectin; (3) factors in regulating plasma adiponectin concentration; (4) adiponectin and insulin resistance; (5) adiponectin in reducing atherosclerosis and hypertension; (6) adiponectin in suppressing liver fibrosis; and (7) peroxisome proliferator-activated receptor γ (PPARγ) ligands thiazolidinediones in inducing adiponectin expression.

L5 ANSWER 43 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2004:946787 CAPLUS
DOCUMENT NUMBER: 142:107509
TITLE: Adiponectin receptors
AUTHOR(S): Yamauchi, Toshiasa; Kadowaki, Takashi
CORPORATE SOURCE: Dep. of Metabolic Diseases, Graduate School of Medicine, University of Tokyo, Japan
SOURCE: BIO Clinica (2004), 19(7), 494-500
CODEN: BCILCY; ISSN: 0919-8237
PUBLISHER: Hokuryukan
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese
AB A review, on adiponectin and receptors, discussing the mechanism of obesity-induced insulin resistance; roles of leptin in insulin sensitivity regulation; adiponectin expression in insulin-sensitive, PPARγ-heterodeficient mouse; adiponectin gene as the pathogenic gene for type 2 diabetes in Japanese; adiponectin as the insulin-sensitive hormone from white adipocytes; adiponectin deficiency and insulin resistance in obesity and type 2 diabetes; roles of adiponectin as antidiabetic and antiatherogenic factors; adiponectin stimulation of fatty acid metabolism; and the identification and functional anal. of adiponectin receptors.

L5 ANSWER 48 OF 54 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2003:333473 CAPLUS
DOCUMENT NUMBER: 139:162642
TITLE: Insulin resistance and role of renin-angiotensin system

AUTHOR(S): Ura, Nobuyuki; Sasaki, Haruki
CORPORATE SOURCE: School of Medicine, Second Dep. of Internal Medicine, Sapporo Medical University, Japan
SOURCE: Bunshi Shin Kekkanbyo (2003), 4(2), 190-196
CODEN: BSKUAB; ISSN: 1345-2355
PUBLISHER: Sentan Igakusha
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese
AB A review on insulin resistance and renin-angiotensin (RA) system discussing (1) RA system activated by insulin resistance, (2) insulin resistance decreased by RA system, (3) increased tumor necrosis factor-α (TNF-α) by RA system, (4) decreased adiponectin by RA system, (5) increased free fatty acid by RA system and (6) increased oxidative stress induced by RA system.

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PASSWORD:
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You either typed them incorrectly, or line noise may
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LOGINID:
SSPTAEGS1646

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NEWS 5	FEB 06	Patent sequence location (PSL) data added to USGENE
NEWS 6	FEB 10	COMPENDEX reloaded and enhanced
NEWS 7	FEB 11	WTEXTILES reloaded and enhanced
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NEWS 9	FEB 19	Increase the precision of your patent queries -- use terms from the IPC Thesaurus, Version 2009.01
NEWS 10	FEB 23	Several formats for image display and print options discontinued in USPATFULL and USPAT2
NEWS 11	FEB 23	MEDLINE now offers more precise author group fields and 2009 MeSH terms
NEWS 12	FEB 23	TOXCENTER updates mirror those of MEDLINE - more precise author group fields and 2009 MeSH terms
NEWS 13	FEB 23	Three million new patent records blast AEROSPACE into STN patent clusters
NEWS 14	FEB 25	USGENE enhanced with patent family and legal status display data from INPADOCDB
NEWS 15	MAR 06	INPADOCDB and INPAFAMDB enhanced with new display formats
NEWS 16	MAR 11	EPFULL backfile enhanced with additional full-text applications and grants
NEWS 17	MAR 11	ESBIOBASE reloaded and enhanced
NEWS 18	MAR 20	CAS databases on STN enhanced with new super role for nanomaterial substances
NEWS 19	MAR 23	CA/CAplus enhanced with more than 250,000 patent

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                equivalents from China
NEWS 20  MAR 30  IMSPATENTS reloaded and enhanced
NEWS 21  APR 03  CAS coverage of exemplified prophetic substances
                 enhanced
NEWS 22  APR 07  STN is raising the limits on saved answers
NEWS 23  APR 24  CA/CAplus now has more comprehensive patent assignee
                 information
NEWS 24  APR 26  USPATFULL and USPAT2 enhanced with patent
                 assignment/reassignment information
NEWS 25  APR 28  CAS patent authority coverage expanded
NEWS 26  APR 28  ENCOMPLIT/ENCOMPLIT2 search fields enhanced
NEWS 27  APR 28  Limits doubled for structure searching in CAS
                 REGISTRY

NEWS EXPRESS JUNE 27 08 CURRENT WINDOWS VERSION IS V8.3,
             AND CURRENT DISCOVER FILE IS DATED 23 JUNE 2008.

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The number of right parentheses in a query must be equal to the
number of left parentheses.

=> S (((Kadowaki, T)/au) OR ((YAMAUCHI,T)/AU) OR ((Kitajima, S)/au) OR ((Ito,
Y)/AU)) AND adiponectin
L1          40 (((KADOWAKI, T)/AU) OR ((YAMAUCHI,T)/AU) OR ((KITAJIMA, S)/AU)
               OR ((ITO, Y)/AU)) AND ADIPONECTIN
```

```
=> Dup rem L1
PROCESSING COMPLETED FOR L1
L2             20 DUP REM L1 (20 DUPLICATES REMOVED)
                  ANSWERS '1-11' FROM FILE MEDLINE
                  ANSWERS '12-14' FROM FILE BIOSIS
                  ANSWERS '15-17' FROM FILE CAPLUS
                  ANSWERS '18-20' FROM FILE EMBASE

=> D Ti L2 1-20

L2   ANSWER 1 OF 20     MEDLINE on STN                  DUPLICATE 1
TI   Adiponectin suppresses colorectal carcinogenesis under the
     high-fat diet condition.

L2   ANSWER 2 OF 20     MEDLINE on STN                  DUPLICATE 2
TI   Adiponectin induces insulin secretion in vitro and in vivo at a
     low glucose concentration.

L2   ANSWER 3 OF 20     MEDLINE on STN                  DUPLICATE 3
TI   Physiological and pathophysiological roles of adiponectin and
     adiponectin receptors in the integrated regulation of metabolic
     and cardiovascular diseases.

L2   ANSWER 4 OF 20     MEDLINE on STN                  DUPLICATE 4
TI   Absence of an association between the polymorphisms in the genes encoding
     adiponectin receptors and type 2 diabetes.

L2   ANSWER 5 OF 20     MEDLINE on STN                  DUPLICATE 5
TI   Hypoadiponectinaemia and high risk of type 2 diabetes are associated with
     adiponectin-encoding (ACDC) gene promoter variants in morbid
     obesity: evidence for a role of ACDC in diabesity.

L2   ANSWER 6 OF 20     MEDLINE on STN                  DUPLICATE 6
TI   Adiponectin stimulates glucose utilization and fatty-acid
     oxidation by activating AMP-activated protein kinase.

L2   ANSWER 7 OF 20     MEDLINE on STN                  DUPLICATE 7
TI   The fat-derived hormone adiponectin reverses insulin resistance
     associated with both lipoatrophy and obesity.

L2   ANSWER 8 OF 20     MEDLINE on STN                  DUPLICATE 8
TI   PPAR gamma agonist and antagonist.

L2   ANSWER 9 OF 20     MEDLINE on STN
TI   Does the -11377 promoter variant of APM1 gene contribute to the genetic
     risk for Type 2 diabetes mellitus in Japanese families?.

L2   ANSWER 10 OF 20     MEDLINE on STN
TI   The mechanisms by which both heterozygous peroxisome
     proliferator-activated receptor gamma (PPARgamma) deficiency and PPARgamma
     agonist improve insulin resistance.

L2   ANSWER 11 OF 20     MEDLINE on STN
TI   The molecular mechanisms by which PPAR gamma/RXR inhibitors improve
     insulin resistance.

L2   ANSWER 12 OF 20  BIOSIS  COPYRIGHT (c) 2009 The Thomson Corporation  on
     STN
TI   Development of high molecular weight (HMW) adiponectin specific
     assay by sandwich ELISA.
```

L2 ANSWER 13 OF 20 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
TI Regulation of bone formation by adiponectin through autocrine/paracrine and endocrine pathways.

L2 ANSWER 14 OF 20 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
TI Effects of Japanese millet protein on lipid profile and diabetes in rats.

L2 ANSWER 15 OF 20 CAPLUS COPYRIGHT 2009 ACS on STN
TI Physiological and pathophysiological roles of adiponectin and adiponectin receptors in the integrated regulation of metabolic and cardiovascular diseases

L2 ANSWER 16 OF 20 CAPLUS COPYRIGHT 2009 ACS on STN
TI Utility of measurements of serum concentration of adiponectin as a diagnostic marker of metabolic syndrome

L2 ANSWER 17 OF 20 CAPLUS COPYRIGHT 2009 ACS on STN
TI Effects of dietary proteins of proso, Japanese and Korean foxtail millets on lipid metabolism and type-2 diabetes in rats and mice

L2 ANSWER 18 OF 20 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI Physiological and pathophysiological roles of adiponectin and adiponectin receptors in the integrated regulation of metabolic and cardiovascular diseases.

L2 ANSWER 19 OF 20 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI PPARγ.

L2 ANSWER 20 OF 20 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI Erratum: Genetic variation in the gene encoding adiponectin is associated with an increased risk of type 2 diabetes in the Japanese population (Diabetes (2002) 51 (536-540).

=> S L2 AND KLF-9
L3 0 L2 AND KLF-9

=> LOg off h
SESSION WILL BE HELD FOR 120 MINUTES
STN INTERNATIONAL SESSION SUSPENDED AT 15:02:07 ON 07 MAY 2009

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:SSPTAEGS1646

PASSWORD:
* * * * * * RECONNECTED TO STN INTERNATIONAL * * * * * *
SESSION RESUMED IN FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE'
AT 15:50:44 ON 07 MAY 2009
FILE 'MEDLINE' ENTERED AT 15:50:44 ON 07 MAY 2009
FILE 'BIOSIS' ENTERED AT 15:50:44 ON 07 MAY 2009
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FILE 'CAPLUS' ENTERED AT 15:50:44 ON 07 MAY 2009
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COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                    27.95      28.17

=> S (Gene therapy) AND Predictab? AND Adiponectin
L4            0 (GENE THERAPY) AND PREDICTAB? AND ADIPONECTIN

=> S (Gene therapy) AND Predictab?
L5          224 (GENE THERAPY) AND PREDICTAB?

=> S L5 AND Review
L6           55 L5 AND REVIEW

=> Dup rem L6
PROCESSING COMPLETED FOR L6
L7            41 DUP REM L6 (14 DUPLICATES REMOVED)
                 ANSWERS '1-7' FROM FILE MEDLINE
                 ANSWERS '8-21' FROM FILE CAPLUS
                 ANSWERS '22-41' FROM FILE EMBASE

=> D Ti L7 1-41

L7   ANSWER 1 OF 41     MEDLINE on STN                  DUPLICATE 2
TI   Current strategies and future directions for eluding adenoviral vector
     immunity.

L7   ANSWER 2 OF 41     MEDLINE on STN                  DUPLICATE 3
TI   Neurological manifestations in lysosomal storage disorders - from
     pathology to first therapeutic possibilities.

L7   ANSWER 3 OF 41     MEDLINE on STN                  DUPLICATE 4
TI   Genetics and molecular biology of chronic lymphocytic leukemia.

L7   ANSWER 4 OF 41     MEDLINE on STN                  DUPLICATE 7
TI   Artificial chromosomes: ideal vectors?.

L7   ANSWER 5 OF 41     MEDLINE on STN                  DUPLICATE 10
TI   The molecular genetic revolution. Its impact on clinical neurology.

L7   ANSWER 6 OF 41     MEDLINE on STN
TI   Current challenges of gene therapy for prostate
     cancer.

L7   ANSWER 7 OF 41     MEDLINE on STN
TI   When and how to assess fast-changing technologies: a comparative study of
     medical applications of four generic technologies.

L7   ANSWER 8 OF 41  CAPLUS  COPYRIGHT 2009 ACS on STN DUPLICATE 1
TI   Gene therapy in Lung transplantation

L7   ANSWER 9 OF 41  CAPLUS  COPYRIGHT 2009 ACS on STN DUPLICATE 5
TI   The use of retroviral vectors for gene therapy-what
     are the risks? A review of retroviral pathogenesis and its
     relevance to retroviral vector-mediated gene delivery

L7   ANSWER 10 OF 41  CAPLUS  COPYRIGHT 2009 ACS on STN DUPLICATE 6
TI   What can SV40-derived vectors do for gene therapy?
```

L7 ANSWER 11 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN DUPLICATE 8
TI New approaches towards ex vivo and in vivo gene therapy

L7 ANSWER 12 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN DUPLICATE 9
TI Gold, the noble metal and the paradoxes of its toxicology

L7 ANSWER 13 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN
TI Systemic therapeutic gene delivery for cancer: crafting Paris' arrow

L7 ANSWER 14 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN
TI Purification of bionanoparticles

L7 ANSWER 15 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN
TI Layers of opportunity: nanostructured polymer assemblies for the delivery of macromolecular therapeutics

L7 ANSWER 16 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN
TI Production of trans-lentiviral vector with predictable safety

L7 ANSWER 17 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN
TI Prodrug and antedrug: two diametrical approaches in designing safer drugs

L7 ANSWER 18 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN
TI Gene therapy versus protein-based therapy: a matter of pharmacokinetics

L7 ANSWER 19 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN
TI Satellite DNA-based artificial chromosomes for use in gene therapy

L7 ANSWER 20 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN
TI Melatonin involvement in cancer: Methodological considerations

L7 ANSWER 21 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN
TI The biology and clinical uses of blood stem cells

L7 ANSWER 22 OF 41 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI Nonsurgical management of hypertrophic scars: Evidence-based therapies, standard practices, and emerging methods.

L7 ANSWER 23 OF 41 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI Surveillance and management of upper gastrointestinal disease in Familial Adenomatous Polyposis.

L7 ANSWER 24 OF 41 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI Assessing neuroprotection in Parkinson's disease: From the animal models to molecular neuroimaging in vivo.

L7 ANSWER 25 OF 41 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI New targets for neuropathic pain therapeutics.

L7 ANSWER 26 OF 41 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI [Cartilage tissue engineering: State-of-the-art and future approaches]. Ingenierie tissulaire du cartilage: Etat des lieux et perspectives.

L7 ANSWER 27 OF 41 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights

reserved on STN
TI Sperm-mediated gene transfer: Applications and implications.

L7 ANSWER 28 OF 41 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI The mechanisms and managements of hormone-therapy resistance in breast and prostate cancers.

L7 ANSWER 29 OF 41 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI Biologic approaches to articular cartilage surgery: Future trends.

L7 ANSWER 30 OF 41 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI Small interfering RNA for experimental cancer therapy.

L7 ANSWER 31 OF 41 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI Hemodialysis access graft failure: Time to revisit an unmet clinical need?.

L7 ANSWER 32 OF 41 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI Scaffold-based bone engineering by using genetically modified cells.

L7 ANSWER 33 OF 41 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI Bone morphogenetic proteins: Relevance in spine surgery.

L7 ANSWER 34 OF 41 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI Revision strategies for lumbar pseudarthrosis.

L7 ANSWER 35 OF 41 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI Gene transfer: A new dimension in anti-depressant therapy.

L7 ANSWER 36 OF 41 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI Interleukin 10 - A therapeutic cytokine for chronic inflammatory diseases.

L7 ANSWER 37 OF 41 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI [Coronary angioplasty at the dawn of the 21(st) century].
Angioplastie coronaire a l'oree du XXI(e) siecle.

L7 ANSWER 38 OF 41 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI Strategies and recent technologies in drug discovery.

L7 ANSWER 39 OF 41 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI Curing IDDM - prevention, repair or replacement.

L7 ANSWER 40 OF 41 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI Intent and coincidence in pharmaceutical discovery. The impact of biotechnology.

L7 ANSWER 41 OF 41 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
TI Delivering foreign genes into mammalian cells.

```
=> Log Off h
 SESSION WILL BE HELD FOR 120 MINUTES
STN INTERNATIONAL SESSION SUSPENDED AT 15:53:06 ON 07 MAY 2009

Connecting via Winsock to STN



Welcome to STN International!  Enter x:x

LOGINID:SSPTAES1646

PASSWORD:
LOGINID/PASSWORD REJECTED

The loginid and/or password sent to STN were invalid.
You either typed them incorrectly, or line noise may
have corrupted them.

Do you wish to retry the logon?
Enter choice (y/N):
Do you wish to use the same loginid and password?
Enter choice (y/N):
Enter new loginid (or press [Enter] for SSPTAES1646):
Enter new password:

LOGINID:
LOGINID:SSPTAEGS1646

PASSWORD:
PASSWORD:
STNLOGON timed out


Connecting via Winsock to STN



Welcome to STN International!  Enter x:x

LOGINID:SSPTAES1646

PASSWORD:
LOGINID/PASSWORD REJECTED

The loginid and/or password sent to STN were invalid.
You either typed them incorrectly, or line noise may
have corrupted them.

Do you wish to retry the logon?
Enter choice (y/N):

Connecting via Winsock to STN

LOGINID:
SSPTAEGS1646

STNLOGON timed out
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Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:SSPTAEGS1646

PASSWORD:
* * * * * * RECONNECTED TO STN INTERNATIONAL * * * * * *
SESSION RESUMED IN FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE'
AT 16:08:33 ON 07 MAY 2009
FILE 'MEDLINE' ENTERED AT 16:08:33 ON 07 MAY 2009
FILE 'BIOSIS' ENTERED AT 16:08:33 ON 07 MAY 2009
Copyright (c) 2009 The Thomson Corporation
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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	55.40	55.62

=> D Hist

(FILE 'HOME' ENTERED AT 14:55:43 ON 07 MAY 2009)

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 14:56:04 ON 07 MAY 2009
L1 40 S (((KADOWAKI, T)/AU) OR ((YAMAUCHI,T)/AU) OR ((KITAJIMA, S)/AU
L2 20 DUP REM L1 (20 DUPLICATES REMOVED)
L3 0 S L2 AND KLF-9
L4 0 S (GENE THERAPY) AND PREDICTAB? AND ADIPONECTIN
L5 224 S (GENE THERAPY) AND PREDICTAB?
L6 55 S L5 AND REVIEW
L7 41 DUP REM L6 (14 DUPLICATES REMOVED)

=> D IBiB Abs L7 6,7,11,13,16,18,27,38,41

L7 ANSWER 6 OF 41 MEDLINE on STN
ACCESSION NUMBER: 1997333815 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9189941
TITLE: Current challenges of gene therapy for prostate cancer.
AUTHOR: Harrison G S; Glode L M
CORPORATE SOURCE: Department of Medicine, School of Medicine, University of Colorado, Health Sciences Center, Denver, USA.
SOURCE: Oncology (Williston Park, N.Y.), (1997 Jun) Vol. 11, No. 6, pp. 845-50, 856; discussion 856-8, 861. Ref: 40
Journal code: 8712059. ISSN: 0890-9091.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199708
ENTRY DATE: Entered STN: 13 Aug 1997
Last Updated on STN: 13 Aug 1997
Entered Medline: 7 Aug 1997

AB Gene therapy for prostate cancer faces hurdles similar to those being encountered for other cancers and nonmalignant processes. The greatest obstacle is the identification of efficient delivery systems, since numerous animal models and cell culture systems have shown potential efficacy when most cells express the introduced genetic material. Early prostate cancers are easily accessible to gene vector introduction, and the predictable metastatic patterns of this cancer may offer additional advantages for gene therapy. This article reviews gene vectors and gene products, as well as ongoing trials of gene therapy that have recently begun in prostate cancer.

L7 ANSWER 7 OF 41 MEDLINE on STN
ACCESSION NUMBER: 1998144156 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9483162
TITLE: When and how to assess fast-changing technologies: a comparative study of medical applications of four generic technologies.
AUTHOR: Mowatt G; Bower D J; Brebner J A; Cairns J A; Grant A M; McKee L
CORPORATE SOURCE: Department of Management Studies, University of Aberdeen.
SOURCE: Health technology assessment (Winchester, England), (1997) Vol. 1, No. 14, pp. i-vi, 1-149. Ref: 375
Journal code: 9706284. ISSN: 1366-5278.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199803
ENTRY DATE: Entered STN: 19 Mar 1998
Last Updated on STN: 15 Sep 2000
Entered Medline: 10 Mar 1998

AB OBJECTIVES. To try to identify the optimal time at which to start assessing new and fast-evolving health technologies. To provide insight into factors influencing the timing of assessments and the choice of methods for assessing new and fast-changing technologies. HOW THE RESEARCH WAS CONDUCTED. A series of literature reviews were undertaken covering the general principles involved in the timing of health technology assessments (HTAs). Additionally, the reported assessments of laparoscopic cholecystectomy, chorionic villus sampling (CVS), teleradiology, teledermatology, genetic screening for predisposition to breast cancer, and gene therapy for cystic fibrosis were reviewed to try to identify the factors that influenced the timing of these assessments. Key individuals in each field were also interviewed. The selected technologies allowed comparison between those that were new and evolving and those that were relatively well-established. A bibliometric study of publication trends was also undertaken to see whether these trends would suggest points in the development of a technology that could be used as indicators that assessment should be started. RESEARCH FINDINGS. TIMING. The precise point at which assessment should start was not identified but the bibliometric study suggested that extending this approach might give useful results. For all health technologies, more regular reporting of outcomes and side-effects should be encouraged during the period after initial assessment and, where the technology is fast-changing, reassessment should take place from time to time. The precise intervals were not identified and the problem remains of deciding when a technology has changed enough to warrant reassessment. FACTORS INFLUENCING TIMING. Published reports of assessments did not generally specify the reasons for their timing, but a number of factors appear to have influenced the timing

of those assessments, directly or indirectly. Product champions and opinion leaders pioneer the introduction of new technologies into clinical practice, and their reports may lead to the rapid diffusion of such technologies before they have been adequately evaluated, as was the case with laparoscopic cholecystectomy; this diffusion may limit the methods of evaluation that can then be used. It is therefore important to assess new health technologies before diffusion takes place. The extent to which regulatory control is imposed on the introduction of new health technologies can also influence the timing of assessments. Such controls might have helped to restrict the diffusion of laparoscopic cholecystectomy, making a large and widely generalisable randomised controlled trial (RCT) feasible. The source and availability of funding for studies may influence the nature and timing of trials. Many telemedicine evaluations were funded by commercial telecommunications organisations and were thus restricted in their timing (and biased towards the technological aspects of the applications) by the availability of funds. Media coverage undoubtedly has an influence although this influence is not always predictable; it may generate 'favourable' publicity about new health technologies, which can lead to immediate demands for the new technique, as was the case with laparosocpic cholecystectomy with its apparent benefits. Thus assessments should be made before media coverage exerts popular pressure on purchasers to adopt the technology and dissuades patients from participating in RCTs (because of fear they may be randomised to the standard treatment as occurred in a US trial of CVS). Innovators should also be cautious in the claims that they make to the media.(ABSTRACT TRUNCATED)

L7 ANSWER 11 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN DUPLICATE 8

ACCESSION NUMBER: 2000:704363 CAPLUS
DOCUMENT NUMBER: 134:202330
TITLE: New approaches towards ex vivo and in vivo gene therapy
AUTHOR(S): Hauser, H.; Spitzer, D.; Verhoeyen, E.; Unsinger, J.; Wirth, D.
CORPORATE SOURCE: Department of Gene Regulation and Differentiation, GBF-National Research Institute for Biotechnology, Braunschweig, D-38124, Germany
SOURCE: Cells Tissues Organs (2000), 167(2-3), 75-80
CODEN: CTORFB; ISSN: 1422-6405
PUBLISHER: S. Karger AG
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review with 11 refs. A number of hurdles have to be overcome in order to achieve efficient and specific gene therapy. Here, two different strategies are discussed that should lead to an improvement of current protocols. A strategy is presented to tag unique chromosomal integration sites by means of retroviral infection, which can be reused for exchange with the gene of interest by action of site-specific recombinases. Targeting exchange is achieved in one step with 100% efficiency by a stringent pos. selection, which makes further screening superfluous. With this strategy a predictable gene expression is obtained for foreign genes integrated into a predefined chromatin structure. A 2nd approach aims at the stabilization of mouse retroviruses towards human serum which is a prerequisite for in vivo gene therapy protocols. To stabilize murine leukemia virus-based retroviruses against human serum, complement regulatory proteins are fused to the retroviral ENV proteins. This results in infectious and human complement-protected particles.

REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 13 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2009:464325 CAPLUS
DOCUMENT NUMBER: 150:365254
TITLE: Systemic therapeutic gene delivery for cancer: crafting Paris' arrow
AUTHOR(S): Tong, Alex W.; Jay, Chris M.; Senzer, Neil; Maples, Phillip B.; Nemunaitis, John
CORPORATE SOURCE: Gradalis, Inc., Dallas, TX, USA
SOURCE: Current Gene Therapy (2009), 9(1), 45-60
CODEN: CGTUAH; ISSN: 1566-5232
PUBLISHER: Bentham Science Publishers Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. Tremendous strides were made in proteogenomics and RNA interference technologies. Hence "personalized" cancer gene therapy has become a foreseeable rather than a predictable reality. Currently, the lack of an optimized, systemic gene delivery vehicle remains a key limiting factor for developing effective treatment applications. Since their introduction by Felgner in 1987, cationic lipids were an attractive consideration for gene delivery, in view of their biocompatibility, biodegradability, low toxicity, and low immunogenicity. Successful in vivo transgene expression by cationic lipid- or cationic polymer-based delivery depends critically on a long circulating half life (>48 h), a definable systemic biodistribution with target-specific cancer localization, and efficient cell entry and internalization. Ideally, the agent should have a hydrophobic, stabilized core that ensures integrity of the therapeutic entity in vivo, a biocompatible, neutrally charged shell (ζ potential of .apprx. ±10 mv) for enhanced, "stealth" circulation, and a suitable size (.apprx.50-200 nm in diameter) for access into the tumor neovasculature and reduced reticuloendothelial system (RES) uptake. "Smart" receptor-targeting moieties can redirect intracellular trafficking. Addnl. engineered features were also incorporated to minimize lysosomal degradation (membrane fusogenic lipids or proton sponge), promote endosomal escape into cytoplasm (cell penetrating peptides, triblock copolymer construction), and enhance nuclear entry and activate the endogenous transcriptional machinery (inclusion of a nuclear localization signal). Improvements in each of these resp. areas of study have converged to yield promising in vivo results.

REFERENCE COUNT: 190 THERE ARE 190 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 16 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2002:850674 CAPLUS
DOCUMENT NUMBER: 138:164126
TITLE: Production of trans-lentiviral vector with predictable safety
AUTHOR(S): Kappes, John C.; Wu, Xiaoyun; Wakefield, John K.
CORPORATE SOURCE: Departments of Medicine and Microbiology, University of Alabama at Birmingham, Birmingham, AL, USA
SOURCE: Methods in Molecular Medicine (2003), 76(Viral Vectors for Gene Therapy), 449-465
CODEN: MMMEFN
PUBLISHER: Humana Press Inc.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. Lentiviruses, with their ability to integrate into the genome of their host, afford unique advantages as vectors for gene transfer. The principal concern with lentiviral vectors, particularly for human gene therapy, is the possibility that replication-competent lentivirus could emerge through genetic recombination. Thus, the burden has been placed on the vector design

itself to ensure the greatest level of safety that is achievable. Authors describe the design of trans-lentiviral vectors that have predictable safety and provide protocols for their production

REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 18 OF 41 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2001:538496 CAPLUS

DOCUMENT NUMBER: 136:349918

TITLE: Gene therapy versus protein-based therapy: a matter of pharmacokinetics

AUTHOR(S): Post, M. J.; Simons, M.

CORPORATE SOURCE: Angiogenesis Research Center, Dartmouth Medical School, Hanover, NH, USA

SOURCE: Drug Discovery Today (2001), 6(15), 769-770
CODEN: DDTOFS; ISSN: 1359-6446

PUBLISHER: Elsevier Science Ltd.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. Gene therapy for pro-angiogenic and antiangiogenic therapy is attractive for several reasons. Conceptually, there is no essential difference between gene therapy and protein therapy, as it is the goal of gene transfer to stimulate transduced cells to locally produce and secrete the desired proteins. However, the two major claims of gene therapy , local delivery and favorable pharmacokinetics, need further consideration. The long-term expression of transduced genes, although heavily dependent on the viral construct, is the major potential advantage over protein therapy. The length of exposure to an angiogenic protein required for its effectiveness has not been established but is widely assumed to be at least a week, a time frame ideally achieved with adenoviral constructs. Slow-release biocompatible polymers have been developed to achieve the same goal for protein therapy. Recent advances in drug-coated stents and injectable biodegradable matrixes can make this option even more appealing. In contrast to predictable pharmacokinetics achieved using these protein-based approaches, viral gene therapies carry a high inter-individual variability in gene expressions. This variation is explained, in part, by variable quantities of circulating anti-adenoviral antibodies and by other, poorly understood parameters regulating cellular and humoral host immune responses to adenoviruses. The same factors also lead to the uncertain duration of gene expression. Thus, the differences between gene and protein approaches for therapeutic angiogenesis are primarily of a practical nature and mundane, nonscientific arguments are likely to become decisive in the near-term.

REFERENCE COUNT: 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 27 OF 41 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2005253239 EMBASE

TITLE: Sperm-mediated gene transfer: Applications and implications.

AUTHOR: Smith, Kevin (correspondence)

CORPORATE SOURCE: School of Contemporary Sciences, University of Abertay, Dundee DD1 1HG, United Kingdom. k.smith@tay.ac.uk

AUTHOR: Spadafora, Corrado

CORPORATE SOURCE: Istituto Superiore di Sanita, Viale Regina Elena, Rome, Italy.

SOURCE: BioEssays, (May 2005) Vol. 27, No. 5, pp. 551-562.
Refs: 116
ISSN: 0265-9247 CODEN: BIOEEJ

COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 022 Human Genetics
029 Clinical and Experimental Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 23 Jun 2005
Last Updated on STN: 23 Jun 2005

AB Recent developments in studies of sperm-mediated gene transfer (SMGT) now provide solid ground for the notion that sperm cells can act as vectors for exogenous genetic sequences. A substantive body of evidence indicates that SMGT is potentially useable in animal transgenesis, but also suggests that the final fate of the exogenous sequences transferred by sperm is not always predictable. The analysis of SMGT-derived offspring has shown the existence of integrated foreign sequences in some cases, while in others stable modifications of the genome are difficult to detect. The appearance of SMGT-derived modified offspring on the one hand and, on the other hand, the rarity of actual modification of the genome, suggest inheritance as extrachromosomal structures. Several specific factors have been identified that mediate distinct steps in SMGT. Among those, a prominent role is played by an endogenous reverse transcriptase of retrotransposon origin. Mature spermatozoa are naturally protected against the intrusion of foreign nucleic acid molecules; however, particular environmental conditions, such as those occurring during human assisted reproduction, can abolish this protection. The possibility that sperm cells under these conditions carry genetic sequences affecting the integrity or identity of the host genome should be critically considered. These considerations further suggest the possibility that SMGT events may occasionally take place in nature, with profound implications for evolutionary processes. .COPYRGT. 2005 Wiley Periodicals, Inc.

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AB In rational drug design, several critical assumptions are made. First of all, the analogs within a series are supposed to act via the same biological mechanism, a precondition which sometimes is not fulfilled. Isosteric replacement of atoms or groups is performed with the expectation that the resulting effects are more or less obvious. Structural variation of the thermolysin and purine nucleoside phosphorylase inhibitors shows that isosteric replacement of atoms may have a significant but hardly predictable effect on biological activities. In some cases, conformational restrictions may lead to a stabilisation of the bioactive conformation while for other structures some additional energy may be required to adopt such a conformation. For ligands causing an allosteric effect, like receptor agonists, biological activity cannot be expected to be a simple function of the binding affinity. And, last but not least, the overall affinity of a drug is by no means only a function of its enthalpic interactions. Pharmacological testing of compounds has shifted

from animal models to in vitro models. Whereas there are unquestionable advantages of this development, there arise also three main problems. Many diseases have multifactorial causes which cannot be tested in a simple in vitro system. Absorption, distribution, metabolism, and excretion of drug candidates are investigated for only few compounds and thus, structural optimization most often neglects these factors. Some side effects of drugs can only be obtained in whole animals, not in in vitro test models. Overall, ligand design is not identical with drug design. Many companies have learned their painful lessons in this direction. To avoid such problems, increasing efforts are made to develop simple screening tests for bioavailability, e.g. cell culture models for intestinal absorption and blood-brain barrier permeation, to use microsomal and liver cell preparations for the investigation of drug metabolism in different species, and to predict toxic effects from short term toxicity models. In the last years, the paradigms of drug discovery changed significantly. Due to the high degree of interdisciplinarity, involving chemistry, molecular biology, biochemistry, pharmacology, and medicine, drug research is almost exclusively performed in industry. Even small venture capital companies, who could give evidence that this kind of research can also be done in a university-like environment, either grow to large companies (like Genentech, Amgen, Affymax, Agouron, and Vertex) or they are absorbed by other companies. Today, pharmaceutical industry reacts very fast to new developments. All major pharmaceutical companies, worldwide, have already shifted or are going to shift a larger part of their capacities from classical syntheses to the synthesis of large libraries of compounds, from classical design to structure- and computer-assisted methods, from in vivo and in vitro screening to faster and faster, fully automated high-throughput screening. Cooperations and mergers of large and small companies lead to a concentration in drug research which will continue in the future. Despite of the enormous efforts of drug companies, there has been a steady decline in the number of drugs introduced into human therapy, from about 60 to 70 new chemical entities (NCE) per year, in the decade between 1970 and 1979, to about 50 NCE's per year, between 1980 and 1989, and to 40-45 in the years 1990-1994. In parallel, the costs of drug research and development increased to about 300-350 Mio US-$ per new drug. Every additional year of drug development is a waste of resources, the money being spent as well as the money being lost due to late marketing. Thus, also time becomes a most important factor in drug development. As the first clinical trials decide on the potential of a new drug, most companies try to arrive at this stage much faster than before. Several drug candidates are developed in parallel, in order to avoid the failure of a whole program if a single compound gives a negative result in its first application to humans. Phase II trials are more carefully planned, to avoid failures in phase III, the most time- and cost-consuming phase in drug development.

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AB In this chapter we have discussed the possible ways in which genes can be delivered into mammalian cells. It is clear that the use of retroviral vectors has been a huge advance in this field. Unfortunately, gene delivery systems are still in their infancy and there is still a long way to go. The potential exists for other viral vector systems and there are other novel strategies for gene delivery now under development. Each vector system has its own problems, but over time it is likely that gene delivery will become more predictable, efficient and effective.

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